REMARKS

Reconsideration of the above-identified application is requested in view of the following remarks.

I. Status of Claims

Claims 1, 3, and 4 are pending in the application. No amendments to the claims are made herein.

II. Rejection Under 35 U.S.C. § 112, First Paragraph (Enablement)

Claims 1, 3, and 4 were rejected under 35 U.S.C. § 112, first paragraph, because the specification allegedly does not provide enablement for a method of preventing an increase in the blood level of IFN-γ in a subject using an interferon-tau having 90% sequence identity to SEQ ID NO: 2.

Analysis

The sole remaining rejection in the application relates to enablement for a treatment method involving an interferon-tau having 90% sequence identity to SEQ ID NO: 2. The Examiner indicated that Applicants can overcome the rejection by showing evidence that the antiviral and conserved regions of interferon-tau disclosed in certain references of record (*e.g.*, Radhakrishnan *et al.* (1999) *J. Mol. Biol.* 286:151-62 and Pontzer *et al.* (1994) *J. Interferon Res.* 14:133-41) are responsible for the reduction in IFN-γ in MS patients (Advisory Action of March 26, 2008).

Applicants traverse the rejection for the following reasons.

1. The antiviral and conserved regions of interferon-tau are known

Applicants submit that when the present application was filed, the antiviral and conserved regions of interferon-tau were known. For example, Pontzer *et al.* provide data that correlate the structure of ovine interferon-tau with regions that exhibit antiviral activity, showing that both interferon-tau and interferon-alpha interact with the type I interferon receptor (page 136, left column), and that peptide fragments of interferon-tau can compete with interferon-alpha interactions with the receptor. In particular, peptides

corresponding to ovine interferon-tau amino acid residues 62-92, amino acid residues 119-150, and residues 139-172, all competed with interferon-alpha, suggesting that they were able to interact with the type I interferon receptor. The authors conclude that these peptides correspond to three antiviral regions of ovine interferon-tau (page 139, left column). In contrast, two peptide fragments, corresponding to residues 34-64 and 90-122, did not compete with interferon-alpha (Fig. 1 and page 135), and antisera generated against the residues 34-64 and 90-122 were not able to reduce interferon-tau antiviral activity (page 136, left column).

Based on such evidence, one of skill in the art would appreciate the areas of the peptide that may be modified without disturbing the activity of the polypeptide and could design polypeptides with 90% sequence identity to SEQ ID NO: 2 that possess therapeutic activity.

Furthermore, Radhakrishnan *et al.* disclose the crystal structure of interferon-tau, which includes five alpha helices and four loop domains that join these helices (page 153, left column). Radhakrishnan *et al.* also disclose a variety of single amino acids that play a role in forming the overall ovine interferon-tau structure, including (in order) Cys1, Leu7, Asn14, Asn22, Arg23, Leu24, Ser25, Asp35, Gly37, Leu38, Gln40, Phe54, Leu57, Ser64, Tyr69, Glu71, His72, Trp77, Cys86, Gln92, Leu96, Cys99, Tyr123, Gly135, Tyr136, Ser137, Cys139, Ala140, Glu142, Arg145, Glu147, Ser151, Ser155, Thr156, Gln159, and Leu162. Based on such evidence, one skilled in the art could make selective mutations in SEQ ID NO: 2 that preserve the helical folding and therapeutic activity.

Thus, based on evidence as provided in, *e.g.*, Pontzer *et al.* and Radhakrishnan *et al.*, one skilled in the art would be able to identify polypeptides having at least 90% sequence identity to SEQ ID NO: 2, and which retain biological activity.

The prior observations by Pontzer *et al.* involving the regions of interferon-tau that bind to its receptor, and the amino acid residues identified by Radhakrishnan *et al.* that are responsible for preservation of the helical structure of interferon-tau, are directly applicable to a skilled artisan making modifications to interferon-tau for use according to the claimed method. Therefore, to practice the claimed method, no additional structure-function information beyond that which is disclosed in the present

application and in references such as Pontzer *et al.* and Radhakrishnan *et al.* is required.

For at least these reasons, Applicants submit that the skilled artisan would be able to identify interferon-tau polypeptide having 90% identity to SEQ ID NO: 2, and to use these polypeptides in the claimed method without undue experimentation.

2. Claims with similar language allowed in a co-pending application

Applicants note that similar claims relating to a method of treatment involving an interferon-tau polypeptide have recently been allowed in co-pending U.S. Application Serial No. 11/112,369 (herein, "the '369 application"). The allowed independent claim in the '369 application reads as follows (emphasis added):[1]

> 1. A method for increasing IL-10 levels in a patient with multiple sclerosis comprising identifying a human subject afflicted with multiple sclerosis; and administering an interferon-tau having greater than about 90% sequence identity to SEQ ID NO:1 in an amount effective to increase blood IL-10 level relative to the blood IL-10 level before administering interferon-tau.

This claim is similar to claim 1 of the present application in that it involves a method of treatment in a subject comprising the administration of an interferon-tau polypeptide having 90% identity to a reference SEQ ID NO. Note that SEQ ID NO: 1 in the '369 application corresponds to the amino acid sequence of mature ovine interferon-tau, which further corresponds to SEQ ID NO: 2 of the present application.

Applicants submit that the present method of decreasing IFN-γ blood levels in a subject with an elevated IFN-γ blood level due to administration of a therapeutic agent for treating multiple sclerosis, and the method for increasing IL-10 levels in a patient with multiple sclerosis claimed in the '369 application, require similar levels of knowledge of interferon-tau structure-function. Moreover, the specification of the '369 application and the present application are similar with respect to their description of interferon-tau. Even the Examiner is the same in both cases. Accordingly, allowance

[1] Note that the reproduced claim reflects the Examiner's amendment proposed in the Notice of Allowance mailed on June 30, 2008.

of the present claims seems reasonable in view of the allowance of the claims in the '369 application, since the language in question is the same.

For this additional reason, Applicants respectfully request withdrawal of the enablement rejection.

III. Conclusion

Applicants believe that the present claims comply with the requirements of 35 U.S.C. § 112. A Notice of Allowance is, therefore, respectfully requested. If the Examiner has any questions or believes a telephone conference would expedite prosecution of this application, the Examiner is encouraged to call the undersigned at (650) 590-0700.

Respectfully submitted,
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